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Syphilis, with Reference to Hereditament Diagnosis and Prevalence.

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In my University teaching and in the daily work of the Pathological Department of the Melbourne Hospital, I have for many years laid stress on the vast importance of inherited syphilis in relation to child mortality, and on certain anatomical signs of syphilis which are very frequent though seldom recognised. Many of my colleagues and friends formerly looked upon me as suffering from a mild monomania, but while growing experience has made my own opinions more precise, a number of accurate observers have been checking my statements in the Melbourne and Children's Hospitals, as well as in forensic work and in private practice. Nevertheless a *resumé* presented to a post-graduate class at the end of 1903 excited much debate, and though I can now appeal to the observations and results of many members of this Society, though some have more or less fully adopted my reading of unquestionable facts, some hesitate and others dissent. And yet I have but been re-discovering or rather emphasising afresh and in detail the phenomena underlying the general statements of Fournier, the prince of modern syphilographers, which have been available even for English readers since 1881. I propose this evening to submit in summary fashion my experience and my beliefs concerning inherited syphilis, and to urge the adoption by practitioners of the marriage-law laid down by Fournier for the guidance of syphilitics. Concerning diagnosis, time will not permit more than a brief statement of my general conclusions, with bald reference to illus-

trative specimens in the University Museum of Pathology (U.M.) As to prevalence, an appendix will contain in tabular form a *précis* of one hundred successive autopsies performed and recorded by me in the Melbourne Hospital. If the Society will honour me by visiting the Museum at the Medical School on some early evening, I will endeavour, with the assistance of some kind friends, to demonstrate the main facts to which I now invite your attention.

Syphilis the Chief Cause of Placental Disease.

Apart from malformation, malposition and accident, syphilis is the principal cause of disease in the placenta. The lesions may be local or general. The arteries are first affected, showing either patchy change allied to atheroma, or widespread thickening and sclerosis, even obliterating the lumina. Severe local change may induce thrombosis with areas of infarction and hæmorrhage (U.M. 5060, 5061), which, when pregnancy is not abruptly terminated, many undergo fibrosis, contraction and scarring (U.M. 5067). Several such specimens have been sent to me in succession from the same patient. When the vascular changes are more general, the whole placenta or large tracts of it may undergo a white fibrosis, often complicated by local thrombosis and hæmorrhage. Such white fibrosis may be seen while the placenta is still small (U.M. 5063—5066), or may be found in one of full size (U.M. 5069 A). In other cases, definite pale gummatous nodes appear (U.M. 5068 A), perhaps with soft mucoid centres (U.M. 5068), either with or without general fibrosis. When the disease is far advanced, earthy salts may be deposited abundantly in the placental tissues on the uterine aspect (U.M. 5069). The fibroid process, even when patchy, often renders the placenta unduly adherent (U.M. 5084), and, if severe and progressive, may be attended with such marked symptoms as to bring the wisdom of complete hysterectomy into consideration (U.M. 5069 A). It is important to remember that gross disease of the placenta is rare, and that in most cases of inherited syphilis the condition of the placenta does not attract attention. The vessels of the umbilical cord may show similar changes, with great thickening of their walls, redness and swelling of the cord, and perhaps thrombosis (U.M. 5069).

Syphilis in Relation to Abortion, Miscarriage and Premature Labour.

Syphilis is one of the most potent causes of interrupted pregnancy, and may operate either by its direct lethal effects on the fœtus or through lesions in the placenta. Death of the fœtus need not be followed at once by expulsion. The redness and maceration of the retained syphilitic fœtus were long ago described by Casper, but these characters depend on post-mortem changes and are not peculiar to syphilis. Extensive disease of the placenta usually causes death of the fœtus, but is compatible with delivery of a living child. The termination of pregnancy by syphilis may occur at any stage, but is most common from the fourth to the sixth month. Repeated abortions and miscarriages are in a large proportion of cases due to syphilis, but may occur in its absence. A few minutes spent in examining the liver of a dead child will probably decide the question, and proper treatment of the mother may prevent any recurrence of mishap.

At inquests on still-born children or on those dying very shortly after birth, evidences of syphilis are present in a large proportion of cases. Several members of the Society, with large forensic experience, would be able to speak on this point with greater precision than I can command. Skin eruptions such as pemphigus have been described in the newly born, but are usually due to maceration. Cases showing genuine eruptions at the time of birth are very rare. As a rule, the signs must be found in the internal organs, and do not differ from those seen in young children, which will be described in detail. In very rare cases, the organs seem healthy, except for the presence of marked and widespread endarteritis.

Paternal and Maternal Inheritance.

On this part of the subject, I desire to draw attention to the statements of the school of Fournier, as represented by Balzer of the Hôpital St. Louis in the Traité de Médecine of Brouardel et Gilbert (1903). Inheritance may come from the father or mother or both. Communication from the infected mother is more sure and more fatal than from the infected father, and not much less sure nor much less fatal than when both parents are

infected. The figures of Fournier as given by Balzer are as follow :—

—	Morbidity.	Fatality.
Both Parents ...	92 per cent.	68·5 per cent.
Mother only ...	84 per cent.	60 per cent.
Father only ...	37 per cent.	28 per cent.

In the act of conception, syphilis may be inherited from either father or mother, although the sperm does not convey syphilis by inoculation. At any later stage, infection comes from the mother through the membranes or the placenta. If the mother contracts syphilis during pregnancy, the child may escape inheritance if the infection occurs at the fifth month, and always escapes inheritance, and therefore has no immunity, if infection is as late as the eighth month. In direct inheritance from the father, Balzer holds that, subject to some very rare exceptions, the mother is always infected, even though no outward sign is shown. In the few exceptions, the mother may *possibly* acquire immunity through the simple absorption of toxins from the infected uterine contents. But Balzer states, on the authority of Rollet, Lewin and Vidal, that a woman, who has borne infected children while herself apparently remaining free, may after re-marriage bear infected children to a clean husband. Cases are on record in which such a woman has after long lapse of time developed tertiary lesions. Apart from such latent infection, on which the law of Colles mainly depends, the mother may contract conceptional syphilis direct from the infected ovum or fœtus, or from some placentary focus, the symptoms usually appearing between the second and fifth month.

In one of his statistical statements, Fournier gives 341 deaths of offspring for 441 confinements. Of the deaths, all but six occurred before birth or during the first year. Of 103 catastrophic confinements, with the father infected and the mother apparently healthy, he found 19 children inheriting the paternal disease, 41 dead before birth, and 43 dead soon after birth.

Signs of Syphilis in Young Infants.

It is well known that the victims of inherited syphilis seldom present definite outward signs of the disease until about the sixth week after birth, when snuffles set in, followed by various rashes.

Suspicion however arises when the child is small and wizened, with dull earthy complexion and loose flabby skin, and confirmation may be given in some rare cases by small mucous tubercles about the mouth or anus. In some instances years pass without unmistakable surface stigmata, and the twelfth year is given as about an average for the delayed expression of inherited syphilis. But even in the absence of any outward sign, the characters of the internal organs are usually distinctive.

The liver is smooth, with thin capsule, the colour varying from shades of pink to greyish or brownish yellow, the cut surface being homogeneous, the lobular structure indistinct, the substance firm and pliant, this alteration in consistence being strongly marked when the organ is cut into thin strips (U.M. 2352). Microscopically there is a fine interlobular cirrhosis, which is usually limited to the small divisions of Glisson's capsule, but which may extend into the lobules in intercellular fashion till the capillaries seem replaced by hyaline or fibrillar growth. Frequently the fine fibrosis is accompanied by fatty infiltration of the hepatic cells, the liver being larger, paler, with less firm consistence (U.M. 2353). Any thickening of the capsule of the liver, or perihepatic adhesions, or gross cirrhosis or gummata must be considered exceptional.

The spleen is rather large, its capsule slightly thick, its substance uniformly firm and pliant (U.M. 2354). The chief microscopic change is an increase in the fine trabeculæ and above all in the network of sustentacular cells.

The kidneys are often lobulated, the capsules peeling without difficulty, the structure seemingly healthy but for some undue pliancy and firmness (U.M. 2357, 2358). Microscopically, there is at first a patchy activity of the nuclei, which are large and stain deeply, especially in the smaller tubes, in the tufts, and in the endothelia of the capillary vessels and lymph spaces. But this early stage is seldom seen, being followed by a thickening of the interstitial tissue of the cortical labyrinth, partly hyaline, partly fibrillar, with multiplying formative cells. The change is still very unequal, so that some tufts are thickly dotted with nuclei of formative cells, while others are little altered.

In the lungs, atelectasis is somewhat frequent ; but apart from this, the lungs are increased in consistence by a fine fibrosis,

partly perivascular and peribronchial, but partly diffuse. The consolidation is slight and general, never complete, never intense. Any patches of bronchopneumonia present are not specific, but due to bacterial complication.

The heart shows ventricular dilatation, which may be strongly marked, and which is usually accompanied by more or less hypertrophy (U.M. 2360, 2358). The valves may be somewhat milky in colour and slightly œdematous, but show no grosser lesions. I do not attach much importance to slight changes in the translucency of the valves, nor to early appearance of opaque patches in the anterior curtain of the mitral valve resembling atheroma. Myocarditis with fibrosis is very rare.

The small arterioles throughout the body show thickening of the coats and activity of the endothelium, endarteritis being sometimes strongly marked when the organs seem fairly healthy (Pavloff). In infants the larger arteries are not notably affected. It is not until about the age of ten or fifteen that precocious atheroma in the aorta, often in delicate linear patterns, furnishes a new sign of the evolution of syphilis.

The changes may be summed up as a fine fibrosis of liver, kidneys, spleen and lungs, accompanied or possibly preceded by thickening of the coats of the small arteries with marked activity of the endothelium. These phenomena bear a strong resemblance to those seen in adults with arteriocapillary fibrosis and fine cirrhosis of the liver and kidney, and must be accompanied with serious autointoxication, due partly to loss of internal secretions, partly to checking of external secretions, partly to general metabolic disorder, and amounting to a condition of instability varying greatly in degree, but comparable with that of latent uræmia.

The severe forms of such disease soon prove fatal, either directly, or by exposing the child without defence to bacterial infection or shock of any kind. But the milder forms persist through childhood, perhaps with little change, perhaps with gradual evolution and increasing intensity. Thus the liver may become more and more firm, perhaps much enlarged (U.M. 2369 *ætate* 11), perhaps with little alteration in size. The kidneys may become more decidedly fibroid (U.M. 2355 *ætate* 6). The slight fibrosis of the lungs may progress till the whole

substance is white, dense and rigid, the cut surface still showing the dominant perivascular and peribronchial mischief, the bronchial glands being similarly affected (U.M. 2380). The heart becomes more and more dilated and hypertrophied (U.M. 2355 *ætate* 6, 2369 *ætate* 11, 2378 *ætate* 20), with increasing arterio-capillary sclerosis, and with incipient atheroma of the large arteries (see Appendix to this paper, Case 45 *ætate* 10, U.M. 2331 *ætate* 17, 2372 *ætate* 22). The lymph glands are often large and hard, with increase of the reticulum and multiplication of formative cells, these changes being sometimes local, sometimes widespread, and causing difficulties in diagnosis from scrofula, tubercle or Hodgkin's disease. Anæmia is always present, and may be profound, even approaching in character to pernicious anæmia. In the milder cases there may be merely a loss of hæmoglobin. But the red corpuscles vary greatly in size, nucleated forms are often abundant, leucocytes are increased to 12,000 or more, and there may be even an approximation to leukæmia (Ewing).

During infancy there is a great tendency to rickets, and it is in syphilitic rickets that we meet the most marked changes in the calvarium, which may be greatly thickened and very vascular, with the characteristic crucial furrow along the sutures (U.M. 2363 A). The anterior ends of the ribs are swollen, with pallor of the new bone next the costal cartilages (U.M. 2363 C). A yellow line of imperfect and degenerate new osseous tissue is found in the long bones at the end of the shaft next to the epiphysial cartilage (U.M. 2363 B). In syphilitic infants, cellulitis near joints is sometimes mistaken for severe rickets (U.M. 2360 *ætate* 2 months).

In these notes, I am avoiding the better known expressions of inherited syphilis, the eruptions, the inflammations of bone, Hutchinson's teeth, interstitial keratitis, etc.

Concerning the widespread fibrosis of organs, on which I have laid so much stress, a cautionary note is necessary. It has already been indicated that such fibrosis may be absent or may be feebly marked in new-born children, though the arteries are widely and gravely diseased (Pavloff). Similarly fibrosis may be absent or feebly marked in children with definite external signs of inherited syphilis; and on the contrary, it may be

strongly marked when the external signs are slight or, for the time being, absent. In other words, transmitted syphilis possesses in strong degree the variable Protean character of the acquired disease.

Syphilis the Great Underlying Cause of Infantile and Child Mortality.

Reference has already been made to the frequency of death shortly after birth in syphilitic children of the worst type. As Fournier says—"These children do not die, properly speaking; they become extinct; they cease to live; for the simple reason that they carry death in their birth; they are unfitted for life by the functional inadequacy of their organs." But he points out that others, though weakly, seem likely to live, but "after a few days, or perhaps a few weeks, they suddenly decline and go off rapidly, without apparent reason, without additional morbid indication. Sometimes . . . they die instantaneously, in a most unexpected and unforeseen manner." Many die in convulsions, and others become hydrocephalic. But Fournier proceeds to state that "in other cases, the children born of a syphilitic father and mother escape both death and the disease" (*i.e.*, the outward phenomena). "But they come with a puny appearance; an impoverished feeble constitution, in a state of persistent anæmia, which resists all remedy; with vital powers much below the average . . . they are often carried away by affections over which they would have easily triumphed had they been more healthy." (Syphilis and Marriage, trans. by Lingard, 1881).

These are the fundamental statements of Fournier, based mainly on clinical experience, and applied by him to cases where the father is diseased and the mother apparently healthy, as well as to cases in which both parents are obviously affected. These are the truths which pathological experience led me to declare in my classes for so many years with increasing insistence.

At this stage I wish to emphasise the poor resistance of these children, even when there is no outward manifestation of syphilis. When a child dies of slight injury, or slight surgical operation, or under chloroform carefully given, or of apparently mild scarlatina, measles, diphtheria, typhoid, pneumonia, influ-

enza, the presumption is in favour of the existence of inherited syphilis. The resistance against colitis, and against pyogenic or tuberculous invasion is also greatly reduced. If we turn to a few Museum speciments, here are organs of the types just described from a boy aged 7 who died under chloroform before an operation for keloid (U.M. 2365) ; of children aged 10, 11 and 12, who died of mild typhoid fever (U.M. 2368 A, 2368, 2367) ; of a child aged 11 dying of mild diphtheria (U.M. 2369). Reference may be made to an infant of 4½ months dying from ulcerative colitis with typical liver, kidneys and heart (U.M. 2363 D) ; to another dying of psoas abscess and multiple cellulitis (U.M. 2364 A—E) ; and to a child dying of disseminated tuberculosis (U.M. 2370). Other cases might be quoted of obstinate anæmia or of intractable œdema (U.M. 2364). In some cases, the tough syphilitic organs are accompanied by gross lesions of mucous membranes, etc., as in a boy aged 5 with extensive scarring at the base of the tongue, and in the pharynx and larynx (U.M. 2361).

But the danger is not limited to childhood. Through pubescence and early adolescence, the same risks continue. If we turn to the Museum again, here are typical organs from a girl aged 17 who died of typhoid fever (U.M. 2331) ; from a girl aged 18 who collapsed after administration of an enema, the autopsy showing general fibrosis of the viscera, blocking of the foramen of Majendie and hydrocephalus (U.M. 2378) ; and from a woman aged 20 with finely granular kidneys, phthisis and dysentery (U.M. 2359).

Later Evolution of Inherited Syphilis in the Organs.

After adolescence, it is difficult to distinguish with certainty between the results of inherited and of acquired syphilis. Many congenital syphilitics with minor degrees of fine fibrosis of the liver and kidneys, with arteriocapillary lesions, with dilatation and hypertrophy of the heart, reach adult life and even pass into its middle and advanced periods. Such persons are more sensible than others to the influence of toxins of all kinds, such as alcohol, and especially to the toxins which underlie the non-syphilitic forms of cirrhosis of the liver, chronic nephritis, pulmonary emphysema, atheroma and aneurism, arteriocapillary

fibrosis and the attendant changes on the heart, local sclerosis and systemic degenerations in the great nervous centres. They are particularly subject to the various forms of anæmia. They may manifest widespread fibrous thickenings of serous membranes and of the associated connective tissues, as in indurative pleuro-pericarditis, chronic mediastinitis, polyorromenitis, etc. As a few examples, I may refer to a case of profound anæmia with fine fibrosis of liver, kidneys, pancreas, etc., in a woman aged 26 (U.M. 2371) ; to commencing lines of atheroma in the descending aorta in a woman aged 22 (U.M. 2372) ; to a case of gummatous and fibrous arteritis with double aneurism in a man aged 52 (U.M. 2339) ; to immense fibrous and gummatous thickening of the pleura and pericardium in a middle-aged man with no previous syphilitic history (U.M. 2379).

The Marriage Law of Syphilis.

As the consequences of inherited syphilis are so grave and widespread, the proper marriage law for syphilitics should be most carefully determined, and rigorously enforced. Opinion has slowly moved since the old time when marriage was considered permissible, if only the secondary eruptions had disappeared. Opinions such as that of Zissl, that syphilis required in most cases only expectant treatment, have died out. But practice is still largely governed in British communities by the dicta of Jonathan Hutchinson, published in 1887. These may be shortly summarised as follow :—" My own rule, for the last twenty years, has been to insist on an interval of two full years between the date of contracting the disease and marriage. . . . I have never relaxed this rule. . . . I am speaking of patients who have been under careful mercurial treatment. . . . Although, however, I have myself had but few opportunities for observation in cases not treated by mercury, yet I am quite prepared to believe that the mere lapse of time is, in most cases, a very efficient cure for syphilis. . . . Probably in a large majority of cases the risk of transmission to children is over long before the end of two years. . . . That tertiary symptoms are not heritable I hold to be well established. . . . I am by no means in a position to deny that the transmission of taint from parent to offspring may appear to continue through

far longer periods. The cases in which it does so are, however, so exceptional, that they need not I think influence our rule . . . a child has much less chance of escape if the mother be diseased than if the disease be confined to the father." In another place he says—"All authorities are agreed that, as a rule, parents who have reached the tertiary stage, although themselves still liable to display symptoms, do not transmit."

More recent observations show that the duration of the secondary or directly infectious period is ill-defined, and that there is an intermediate period in which tertiary phenomena may overlap secondary phenomena. There is also a small but dangerous group of cases in which secondary lesions tend to recur during several years as if by repeated relapse. As to the tertiary period, many authorities believe that *absolute* security from transmission to children is never obtained by mere lapse of time. Thus Whitehead has narrated a case in which a woman had primary syphilis soon after her first marriage at the age of 17. She had slight secondary symptoms for two or three years, but was deemed cured. Her husband died twelve years after the marriage. She married again to a man with no history of syphilis, and bore him a syphilitic child fifteen years after her first marriage. She herself continued in good health.

Fournier's Lectures on Syphilis and Marriage, translated into English in 1881, inculcate a much more stringent marriage law. Jonathan Hutchinson wrote a preface, in which he stated that, while fully agreeing with the reasoning, he could not on the ground of expediency go so far in prohibiting or delaying marriage. Fournier admitted that it was rare for a husband to infect his wife if his syphilis dated three or four years before marriage, and very rare if it dated more than six years back; and that where syphilis passed from the father to the child while the mother showed no sign, the paternal infection never dated back more than three or four years. [Yet on page 262, he records a case with such inheritance fourteen years after infection.] Fournier gives as his conclusion—"I do not believe that it may be permitted to a syphilitic subject to dream of marriage under a *minimum* of *three to four years*, devoted unremittingly to treatment." The treatment should be by mercury and iodides, given in the successive or intermittent fashion, so as to maintain

intensity of action. There should be no sign of syphilis for a period before marriage of eighteen months to two years. Malignant or inveterate forms of syphilis should be an absolute bar to marriage.

These doctrines of Fournier are largely adopted in America. Thus in the students' "Manual of Syphilis," by Hyde and Montgomery of the Rush Medical College, the authors say that "it is impossible to lay down rules for all cases, but the following limits are fairly well established in practice. A previously healthy young man or woman, skilfully treated for between three or four years after infection, and free for the last year from any but the most insignificant symptoms, will in the large majority of cases fail to infect a married partner or transmit syphilis by inheritance. No man should marry, whatever time may have elapsed after infection, who has not had a long interval—at the very least six months—of absolute freedom from symptoms; and the reverse is true, that no man should marry, however remote the date of his infection, who bears upon his person active symptoms of the disease. There are subjects of syphilis who should never marry, though these are few. In them the disease has induced a cachexia permitting an evolution of the malady to the point where the systemic infection is too profound and too persistent to permit a return to a normal standard of health."

The latest authoritative declaration in France is given as follows by Balzer in the Traité de Médecine of Brouardel et Gilbert (1903) :—"In practice we admit, with the majority of authors, that marriage can be authorised in and after the fifth year, but with the fulfilment of the following conditions; specific treatment, sufficient and regularly carried out" [*i.e.*, during four years on the intermittent intensive system]; "no sign of syphilis during the fourth year of the disease. It is prudent to advise in addition a preventive treatment at the time of marriage. It must not be forgotten that maternal heredity lasts longer than paternal. Kassowitz believes that it may show itself during ten years. Hence it is wise to continue preventive treatment in women for a long period."

But there is no reason for pessimism. As Fournier says, "the truth is that, with some very rare exceptions, syphilis only constitutes a temporary bar to marriage; and a syphilitic sub-

ject, after a proper period of probation, regains a state of health which renders him fully capable of the double *rôle* of husband and father."

Diagnosis of Tertiary Syphilitic Lesions.

Time and space compel me to deal but briefly with some selected points of greater or less importance.

General Indications.—In the pathological diagnosis of tertiary syphilitic lesions, the most valuable information is afforded by the small arteries of microscopic size. In the most typical cases, all the coats are thickened, and the lumen may be reduced even to the point of obliteration, or thrombosis may occur, with varying degrees of organisation of the clot. In the slightest cases, there may be simply an undue activity of the endothelium, the cells being swollen with deeply staining nuclei. In more severe cases, the endothelium may be cubical or columnar, perhaps standing up in greatly elongated forms. The muscular coat is usually thickened, with multiplication of muscle cells, which may proceed till the middle coat is enormously thick, the concentric lines of cells at once attracting attention. The outer coat may be greatly increased, with varying degrees of sclerosis, and fibrillar change may radiate from it into the tissues around. The coats frequently suffer unequally, so that one or other may show the most marked changes. Caution is necessary in interpreting the meaning of such changes in the vascular walls, especially in dealing with parts subject to pressure or repeated irritation, such as the sole of the foot, or with polypoid tumours in the throat or in the uterus, or with the tissues of patients suffering from general arteriosclerosis.

Syphilitic growths have several other marked characteristics. They frequently develop rapidly in alarming fashion, and then pass into an indolent or stationary period, and frequently show some signs of repair. If ulceration is present, the epithelial edges are inactive, not fungous, not infiltrating. The base may show the typical wash-leather condition, but varies greatly. The neighbouring lymph glands are often enlarged, but in a chronic painless non-progressive manner. If caseation occurs in these glands, it is usually dry, and the caseous patch is often very sharply defined. Syphilitic growths may proceed to rapid casea-

tion and softening, or may tend to slow fibrosis. In the slow forms, the microscope shows formative cells and fibrillar tissue, at parts staining well, but at parts possibly clouded by imperfect caseation ; but a notable feature is generally presented by scattered foci of recent activity, displaying clusters of small round cells, which may be young formative cells or leucocytes. In syphilitic growths, the endothelia of the lymph spaces are often active, so that various types of syphilitic endothelioma may arise.

In the examination of syphilitic organs, attention is too often limited to such gross lesions as perihepatitis, dense pleural or pericardial adhesions, etc. But valuable information is often given by arborescent opaque markings in the epicardium, chiefly along the coronary grooves and the edges of the heart, or by linear opacities in the pulmonary pleuræ or the serous capsule of the liver, especially along the lines of the larger lymphatics.

In autopsies, the deep surface of the calvarium should always be examined. If it shows abundant deep vascular channeling, and especially if the surface is copiously engraved with very fine grooves, a strong suspicion of syphilis should be entertained. Such grooves reveal the presence of widespread arterial thickening, extending to the minute branches.

Atheroma.—The following forms are, in my opinion, sure indicia of syphilis :—(a) atheroma in young subjects, and in particular the delicate linear forms of atheroma seen in inherited syphilis, especially in the descending aorta (U.M. 2372) ; (b) small round nodules, sharply prominent, often deep yellow, seen chiefly in the ascending aorta (U.M. 2332) ; (c) the intense ulcerous form (U.M. 204 and 2333) ; (d) the late atrophic form, with closely set wrinkles (U.M. 2342) ; (e) large patches of great thickening with glossy surface in the ascending aorta near the valves (U.M. 2338) ; and to these may be added, though with less confidence, (f) cases of intense and widespread atheroma with great thickening (U.M. 2336 and 3967), and (g) cases with evidence of definitely recurrent lesions, fresh white plâques, old yellow patches, wrinkled scars, and possibly ulcers and calcified plates occurring side by side (U.M. 2340 A). In the two last groups, a positive diagnosis should not be made without confirmatory evidence. The above mentioned lesions in the aorta are frequently associated with a peculiar atrophic atheroma or

chronic arteritis of the iliac and other smaller arteries, which I also regard as indicating syphilis. Marked atheroma of the pulmonary artery is nearly always syphilitic..

Valvular Disease of the Heart.—Syphilis is a frequent cause of chronic endocarditis, and especially of mitral stenosis. The syphilitic cases include those with excessive thickening and rigidity, which may be associated with curious festooned opaque markings in the endocardium of the ventricular septum (U.M. 2344). Strongly marked fibroid thickening of the rings at the bases of the valves is in many cases of syphilitic origin. The segments themselves may escape.

Myocarditis.—Nearly all, if not all, chronic indurative myocarditis is due to syphilis (U.M. 3657—3667). The more rapid forms with fleshy thickening are invariably syphilitic (U.M. 3668). Aneurismal dilatation at the left apex is usually a result of syphilitic myocarditis (U.M. 3679—3682).

Syphilis of the Lungs includes (a) many cases or pleurogenic fibrosis, with or without gummata (U.M. 3437—3439) ; (b) cases of fine cirrhosis, independent of dust disease, more or less patchy, the fibrosis radiating chiefly from the arteries and tubes, the intervening tissues emphysematous (U.M. 3436) ; and (c) the white pneumonia of inherited syphilis (U.M. 2380) ; and syphilis cooperates with other factors in (d) chronic pneumonia following upon croupous or catarrhal pneumonia (U.M. 3435 and 3357) ; and (e) syphilitic phthisis, with its dominant fibrosis and foci of chronic encapsuled suppuration (U.M. 3440—3442).

Polyorromenitis, Chronic Mediastinitis, etc.—Syphilis is the underlying cause of most of these affections (U.M. 4200 and 3857) .

Syphilis of the Lips is characterised by its frequent position at the angle of the mouth, by the inactivity of the epithelial edge, by the frequent evidence of repair, and by the fact that on section a chronic inflammatory condition is shown, in which the white fibrocellular infiltration does not completely hide the muscle bundles (U.M. 2275).

Syphilis of the Tongue needs much more careful consideration than it receives. Rapid gummatous destruction is easily distinguished, but chronic deep seated gumma with slight erosion of the surface may easily be confused with epithelioma (U.M. 2277),

if the condition of the epithelium bounding the erosion is not carefully examined. Chronic syphilitic ulcers on the dorsum near the root (U.M. 2381) may easily be confounded with chronic epithelioma (U.M. 1524). Epithelioma in syphilitics may assume peculiar forms.

Syphilis of the Larynx in the form of gummatous thickening (U.M. 2260), or of sharply defined ulcer (U.M. 2257), with the attendant engorgement, may be easily distinguished in nearly all cases from the pale superficial ulceration of tubercle (U.M. 2085), or from the ulcerative or vegetative forms of epithelioma (U.M. 1540 and 1538). But diffuse gummatous thickening may easily be confounded with the diffuse obstructive form of epithelioma (U.M. 1536). Fortunately both are very rare.

Syphilis of the Stomach may appear (a) as a chronic shallow ulcer with sinuous edges, usually at or near the lesser curvature, having a tendency to heal with little cicatricial contraction (U.M. 2284) ; or (b) as a chronic diffuse fibrocellular infiltration of the entire pyloric region, the different layers being thickened and perhaps matted together, but without epithelial infiltration, the surface of the mucous membrane being uneven, with a tendency to slight papillary thickening or superficial ulceration (U.M. 3911) ; or (c) as similar fibrosis more localised near the pylorus, with more defined chronic ulceration of the surface, the ulcer having a hard base and edge, with no activity of the edge, no epithelial infiltration (U.M. 3912, 3912 A). Hardened lymph glands may be present in syphilis or carcinoma.

Syphilis of the Small Intestine is very rare. The Museum has only one specimen with large square ulcers in Peyer's patches, with dense fibroid bases, and inactive firm edges, the peritoneal surface showing no groups nor lines of tubercles, but little fibrous tags indicating very chronic irritation. (U.M. 2285 A).

Syphilis of the Rectum is seen chiefly in the female, and shows three forms which may be combined—(1) Sinuous superficial ulceration (U.M. 2292) ; (2) dense fibrous stricture, with curious pits and bridles above (U.M. 2289) ; and (3) diffuse gummatous thickening and rigidity (U.M. 2287).

Syphilis of the Testis occurs in (a) the rapid gummatous form, imitating sarcoma (U.M. 2311) ; (b) the slow gummatous form with more or less fibrosis (U.M. 2307) ; (c) the fibroid form with-

out gummata (U.M. 2320). The second and third of these forms may occur on opposite sides of the same patient (U.M. 2316). The gummatous form *may* present itself without irregularity of the surface, and the fibroid form with irregularity. The tendency to fungate *may* be absent in rapid cases and present in slow cases.

Syphilis of the Uterus in its larger grosser forms is practically confined to the cervix, and is usually associated with extensive lesion of the upper part of the vagina. It occurs in a rapid form, with soft gummatous destruction of the cervix, vagina and floor of the bladder, too rapid for epithelioma (U.M. 2327) ; and in a slow form with fibrocellular infiltration and contraction, too slow for carcinoma (U.M. 2328). Syphilitic Endometritis is by no means uncommon, and may be attended with troublesome hæmorrhage and excite a suspicion of cancer. Curettings show an even or granular or slightly papillary surface, and microscopically present typical changes in the small arteries. The gland tubes may be active, but their epithelium is well defined, not tending to break loose nor to become atypical. Slight cystiform dilatation may be seen. The matrix is unequally cellular, without the embryonic vessels or the typical cells of sarcoma, and sometimes shows traces of a tendency to organisation.

Syphilis of the Ovaries is rare as a separate disease. Early fibrosis may be seen in inherited syphilis, and may favour the development of cystic disease (U.M. 1694, *ætate* 18). Sclerosis of the ovary from other causes may be aggravated by syphilis.

Syphilis of the Mammary Gland is seldom gummatous, but is usually characterised by chronic inflammatory changes, very indolent, with periods of inaction, but tending towards fibrosis and induration. Cysts may appear. There may be pyogenic complication. Otherwise the skin is not adherent, the nipple usually not retracted, and after a long history the axillary glands *may* be unaffected, and the general health fairly good (U.M. 5137, 5138). Cases, however, may finally pass into carcinoma.

Syphilis of Muscle may appear in rapid soft gummatous growth resembling sarcoma, as in a specimen infiltrating the psoas muscles (U.M. 2233) ; or in a slow form with patchy fibrocellular thickening, whitish areas of dense fibrosis contrasting with younger patches showing formative cells and fibrils, with here

and there recent foci of irritation thickly dotted with small cells even in the midst of the denser tissue. The Museum has a excellent specimen removed from the serratus magnus on th outer wall of the thorax (U.M. 2235). Forms intermediat between these extremes may also occur as in specimens fro the quadriceps extensor cruris (U.M. 2234 and 2234 A). A indicated in connection with syphilis of the lips, the persistenc of voluntary muscle fibres in the midst of a suspicious cellula infiltration is an important sign. The muscle fibres, thoug persistent, may be variously changed, irregular in outlin shrunken, with patches of hyaline change or still more curiou vacuolation. When syphilitic lesions of muscle undergo retro gression and partial resolution, the vessels may dilate so as t form peculiar cystic structures, as exemplified in a specime removed from the pectoral region (U.M. 1670).

Syphilis of Skin.—Time permits me only to refer to smal syphilitic growths which may simulate sarcoma. Removal i easy and makes for safety. The microscope shows an absenc of the definite histologic elements of sarcoma, a patchiness o change with older fibrillar areas dotted with recent groups o small cells, marked activity of the endothelia of lymph space perhaps amounting to endothelioma, and above all the typica lesions of the small arteries.

Syphilis of Bone has been very thoroughly studied. But suffi cient attention has not been given to the influence of syphilis o chronic tubercular and pyogenic processes, in increasing the ten dency to new formation of bone on the surface, either in diffus or stalactite fashion, and the tendency to deep sclerosis. Syphili also seems to aggravate the bony changes in osteoarthritis defor mans, and sometimes to determine an early and severe form o this disease (U.M. 2381).

Syphilis of the Spine is rare. It may reveal itself as carie sicca, the body of one or more vertebræ crumbling away an being absorbed, without suppuration or marked caseation (U.M 3196) ; or it may assume a chronic inflammatory type, wit patches of sclerosis, fusion of the bodies of vertebræ, foci o chronic suppuration, with dense-walled sinuses leading throug the lumbar muscles (U.M. 3197).

Intracranial Syphilis occasions little difficulty in diagnosis when ffecting the dura mater, whether in the form of firm local rowths (U.M. 2238) or in that of diffuse gummatous thickening U.M. 2243). Gummata on the arteries cannot be mistaken U.M. 2247). But difficulties may arise in the diagnosis of liomata from gummata. Some gliomata resemble hypertro-hies, *e.g.*, an overdevelopment of the pons Varolii (U.M. 1066); thers are well-defined and fleshy (U.M. 1071) ; others are firm ith more or less fibrous grain (U.M. 1070). These are easily ecognised. Caseation with central softening is distinctive of yphilis (U.M. 2248). Widespread caseation, even without oftening, is strongly in favour of syphilis (U.M. 2249), though he Museum contains a caseating glioma (U.M. 1061). Exten-ive softening around the growth is more common in syphiloma. Microscopically, the spider cells of glioma and the vascular hanges of syphilis are the chief diagnostic elements.

Prevalence of Syphilis.

In pathological practice I very rarely meet with syphilis in he primary or secondary stage. The lesions are almost always hose of tertiary or of inherited syphilis. An appendix contains n tabular form the analysis of one hundred *consecutive* autopsies erformed and fully recorded by me in the Melbourne Hospital. Of the hundred cases, according to my criteria, 34 showed clear igns of syphilis, 19 others showed doubtful signs, and 1 was pen to suspicion.

The 34 cases that I regard as certain may be classified as ollows :—

Showing gummata or nodes or scars on face	10
Special types of atheroma in young persons under 27, with fibrosis of organs, patchy thickening of serous capsules	6
Extreme ulcerous atheroma, atheroma in all stages, fine fibrosis of organs, patchy thickening of capsule of spleen ...	1
Stricture of rectum, caseating glands, severe atheroma, fine fibrosis of organs, *ætate* 33	1
Fibrosis of lungs, patchy fibrosis in sheath of pulmonary artery and in pleura, slight pulmonary atheroma, *ætate* 32 ...	1
Chronic myocarditis, with fibrosis of organs	2
Special forms of atheroma, with various confirmatory signs ...	13
	34

Of the 19 cases called doubtful, I can only say in general terms that they were somewhat less decided than the foregoing. The suspicious case was a man of 60 with stricture and pyonephrosis, with patchy thickening and opacity of the capsule of the liver, with irregularly atrophic granular kidneys, and slight fibrosis of the liver and spleen.

It is very important to note that only one case presented nodes on the bones, only one case an ulcer in the skin, and only two cases showed a sunken nose or cutaneous scars. Many of the most typical specimens of acquired syphilis in the Museum were obtained from patients with no external sign of the disease.

In estimating the meaning of the present analysis, it must be understood that I examine only a fraction of the cases dying in the Hospital. In my daily work, when there is choice of a case with phthisis or one without, I examine the one without. The general percentage of syphilis would probably be considerably less for the period represented by my analysis. I am also inclined to believe that the incidence of syphilis was unusually heavy in the hundred cases now scrutinised, though the particular hundred was selected in the most casual way. The Melbourne Hospital, too, is the final refuge for a large proportion of the flotsam and jetsam of the metropolis. It never closes its doors against the dying. Hence my figures must be taken for what they are, and no more general meaning must be read into them. They do not apply to the living Hospital patients, they do not apply to the general community. Nevertheless the results of the analysis were more grave than I expected.

A cursory examination of the appendix will also indicate the enormous frequency of fibrous changes in the liver and kidneys even in cases not recorded as even doubtfully syphilitic.

Syphilis is comparatively seldom the direct immediate cause of death. Its baneful influence is hidden behind the names of many common fatal diseases, such as granular kidneys, Bright's disease, nephritis, uræmia, anæmia, dropsy, cirrhosed liver, arteritis, atheroma, aneurism, apoplexy, myocarditis, chronic endocarditis, locomotorataxy, general paralysis, etc. Cases of these diseases need careful post-mortem examination before we can determine, with some degree of probability, which are due to syphilis and which are not. Hence ordinary statistics are almost of no value in estimating the prevalence of syphilis.

APPENDIX.

Analysis of 100 *Consecutive Autopsies performed by Professor Allen.*

No.	Sex	Age	Cause of Death.	Signs of Syphilis.	Atheroma not Typical.	Fibrosis of Organs, and Notes.
1	F	49	Gangrene of legs from arterial thrombosis	—	Slight	Liver, kidneys, spleen, arteriocapillary. Hour-glass stomach, with scar of ulcer.
2	M	17	Sacroiliac disease; Iliopsoas abscess	Linear atheroma in abdominal aorta	—	Fine fibrosis, with lardaceous disease widespread.
3	F	63	Carcinoma of pancreas and liver	Atheroma in all stages. Calcified gumma in spleen	—	Tough subgranular kidneys.
4	M	57	Cirrhosis of liver; arterio capillary fibrosis	—	Slight	Liver, kidneys, spleen, arterio-capillary.
5	F	33	Bronchopneumonia	Stricture of rectum Caseating firm glands along abdominal aorta, in mediastinum and neck	Severe, but not typical	Unilobular cirrhosis of liver. Finely granular kidneys.
6	M	45	Hæmorrhage in pons varolii	Atheroma in all stages. Deep arterial grooves inside the calvarium (?)	—	Liver, spleen, kidneys. Feebly hour-glass stomach. Nodular thickening of capsule of spleen.
7	F	32	Patent ductus arteriosus. Pulmonary fibrosis and bronchiectasis	Fibrosis of lungs Fibroid patch on pericardial sheath of pulmonary artery. Fibroid patches in pulmonary pleura	Slight atheroma of pulmonary artery	Marked in spleen, slight in kidneys. Intense curvature of spine. Advanced osteoarthritis. Hour-glass stomach, with dilated duodenum.
8	M	64	Cerebral apoplexy	—	Severe in aorta and smaller arteries	Severe spondylitis deformans. General emphysema. Firm cystic kidneys.
9	F	65	Chronic bronchitis and emphysema	All stages of atheroma. Linear patches of opacity in epicardium, in pulmonary pleura, and in capsule of liver	—	Cord-like bands of adhesion of liver and stomach to parts around. General emphysema.
10	M	26	Patent foramen ovale, pulmonary obstruction, peripheral systemic obstruction, &c.	Yellow atheroma. Fine fibrosis of liver and spleen. Caseation and calcification of mesenteric glands with no ulceration of intestine. Linear opacities in pericardium	—	Ill-developed, with undescended testes. Sacculi in duodenum Doubtful gliosis in medulla oblongata and cervical cord.
11	M	37	Subarachnoid hæmorrhage in pons	Yellow atheroma with linear tendency	—	Fine fibrosis of liver and spleen. General emphysema. Pliant kidneys, with cysts.
12	F	41	Uræmia. Right kidney atrophied and subgranular	Large area of pliant fibrosis at base of right lung. (No tubercle)	—	Liver and spleen. General emphysema. Epilepsy.
13	M	45	Chronic ulcer of stomach, with secondary carcinoma	Sharply defined yellow elevated patches in narrow lines throughout aorta	—	Fine fibrosis of liver and kidneys. Emphysema.

No.	Sex	Age	Cause of Death.	Signs of Syphilis.	Atheroma not Typical.	Fibrosis of Organs, and Notes.
14	M	36	Aortic obstruction	Yellow atheroma in linear patterns. Opacity of epicardium over coronary vessels. Lines of fibroid opacity in capsule of liver	—	Fine fibrosis of liver and spleen. Fibrosis and calcification of aortic ring and bridles.
15	M	41	Lobar pneumonia	Sharply defined prominent yellow dots of atheroma	—	Fine fibrosis of liver and spleen.
16	M	51	Arteriocapillary sclerosis. Large subgranular kidneys	(?)	Old atheroma of abdominal aorta. Atrophic atheroma and aneurismal dilatation in superior mesenteric (?)	Fine fibrosis of liver, kidneys, and spleen. Emphysema. Patchy fibroid thickening of capsule of spleen. Marked spondylitis deformans.
17	M	70	Aortic endocarditis. Softening of internal capsule of cerebrum from embolism	(?)	Calcified plates in ascending aorta. Large yellow patches in arch and descending aorta. Atrophic wrinkling in iliacs. Probably syphilitic	Nutmeg fibrosis of liver. Subgranular kidneys. Aneurisms of left ventricle below the valves
18	M	42	Post-influenzal croupous pneumonia	—	—	Very slight in liver.
19	F	17	Typhoid fever	Liver and kidneys decidedly firm. Sharply defined prominent yellow specks of atheroma more or less linear	—	Retroverted fallen uterus, with marked anteflexion.
20	M	57	Cirrhosis of liver	—	—	Marked anteroposterior curvature of tibiæ. Unilobular and multilobular cirrhosis of liver. Emphysema.
21	M	26	Glomerulo-nephritis. Uræmia	—	Very slight	Hypertrophied dilated heart.
22	M	39	Bronchopneumonia in a gouty subject		—	Subgranular kidneys. Fibroid tubercle and conglomerates in lungs.
23	M	20	Mitral obstruction	Small yellow patches of old atheroma, and large areas of recent atheroma	—	Very large thymus gland. Fine cirrhosis of liver and kidneys. Old caseation in mediastinal and bronchial glands.
24	M	65	Uræmia	Wide-spread atheroma in all stages. Ulcer on inner malleolus	Atheroma of coronaries	Atrophic fibroid kidneys. Slight fibrosis of liver. Emphysema. Thickening of fibrous rings of aortic and mitral valves with calcification.
25	F	56	Large subgranular kidneys, pulmonary thrombosis, myomalacia cordis	(?)	Large calcified plates in the descending aorta Widespread atheroma in iliacs. Calcified coronary arteries	Nutmeg fibrosis of liver. Vascular grooves in calvarium deep. Cæcum just below liver.
26	F	62	Arteriocapillary sclerosis. Atrophied kidneys. Pulmonary thrombosis	Severe atheroma in various stages with ulceration	Extreme atheroma of the coronary and cerebral arteries	Calvarium deeply grooved. Endothelioma on dura mater. Patchy thickening of capsule of spleen.

No.	Sex	Age	Cause of Death.	Sign of Syphilis.	Atheroma not Typical.	Fibrosis of Organs, and Notes.
27	M	38	Arteriocapillary sclerosis. Dropsy	Severe syphilitic atheroma in all stages, with thrombosis and aneurism. Small yellow thickening in capsule of liver. Calcified nodule in posterior mediastinum	—	Fine fibrosis of kidneys.
28	M	55	Arteriocapillary sclerosis. Dropsy	(?)	Very slight	Long standing suppuration beneath the ribs on right side. Great fibrosis and calcification.
29	M	72	Softening and hæmorrhage in internal capsule	—	Slight in aorta. Patches in carotids and coronaries	Slight nutmeg fibrosis of liver. Subgranular kidneys. Enlarged prostate. Fibrosis in aortic valves.
30	F	30	Tuberculosis of lymph glands and lungs with acute pleurisy and pericarditis	—	—	Doubtful lymphoma and tubercle. Subgranular kidneys.
31	M	24	Pulmonary phthisis	—	—	Lardaceous disease. Milky epicardium.
32	M	54	Pulmonary phthisis	—	—	Fibrofatty liver. Subgranular kidneys. Perforating ulcer of intestine. Spondylitis.
33	F	19	Strangulated umbilical hernia	—	—	Fibrofatty liver. Subgranular kidneys.
34	M	60	Stricture of urethra, pyonephrosis	Patchy thickening and opacity of capsule of liver (??)	—	Slight fibrosis of liver and spleen. Irregular atrophic granular kidneys. Emphysema.
35	M	53	Sarcoma of base of skull. Abscess of brain	—	—	Autopsy not quite complete.
36	F	56	Carcinoma of stomach	—	—	Ulcers in gall-bladder.
37	F	40	Croupous pneumonia (post puerperal)	—	—	Patchy atrophy of kidneys.
38	M	66	Ulceration of stomach, bronchopneumonia with gangrene of lung	Atheroma in all stages. Atrophic atheroma in iliacs.	—	Fine fibrosis of liver. Pliant kidneys. Hard pseudotubercles in pleura. Calcified nodules in mesenteric glands.
39	F	84	Atrophic granular kidneys, &c.	Extreme ulcerous atheroma. Atheroma in all stages. Atrophic wrinkling in iliacs	—	Fine fibrosis of liver. Patchy fibroid thickening of capsule of spleen.
40	M	82	Strangulated hernia, &c.	Atheroma in all stages, with slight ulcers (?)	Severe atheroma in cerebral arteries	Subgranular kidneys. Fine fibrosis of liver. Patchy fibrous thickening of capsule of spleen. Fenestration of foramen ovale.
41	M	60	Pneumonia, pleurisy, pericarditis	Patchy fibrous thickening of capsule of liver (?)	Slight, but widespread	Large pliant kidneys, with atrophic patches.
42	M	64	Diphtheria	—	—	Subgranular kidneys. Emphysema. Slight fibrosis of liver, with fatty infiltration.
43	M	49	Influenza. Toxic ascending paralysis	—	Slight	Deep channels in calvarium.
44	M	23	Pulmonary and intestinal tuberculosis	Atrophic atheroma of iliacs; patchy thickening of capsule of liver	—	Lardaceous disease.

No.	Sex	Age	Cause of Death.	Signs of Syphilis.	Atheroma not Typical.	Fibrosis of Organs, and Notes.
45	F	10	Mild prolonged typhoid fever. Pneumonia	Fine fibrosis of liver, spleen, kidneys. Sharply defined prominent yellow spots of atheroma	—	Peripheral resistance. Hypertrophy and dilatation of heart.
46	F	39	Puerperal thrombosis and septicæmia	Sunken nose ; scars on face	—	Subgranular kidneys.
47	M	52	Carcinoma of stomach	Syphilitic arteritis (?)	Atheroma at roots of coronary arteries	Patchy atrophy of kidneys. Emphysema. Old perihepatitis.
48	F	63	Removal of gasserian ganglion. Bronchopneumonia	—	Slight in aorta and cerebral arteries	Subgranular kidneys. Osteoarthritis. Malformed liver.
49	M	60	Volvulus of pelvic loop	—	Slight	Smooth pliant liver. Large subgranular kidneys. Very large prostate. Emphysema.
50	F	26	Puerperal septicæmia	—	—	—
51	M	58	Granular kidneys. Uræmia	—	Intense in cerebral arteries	Smooth pliant liver. Emphysema. Patchy softening of brain.
52	M	57	Large granular kidneys. Arterio-capillary fibrosis	—	Atheroma of coronary arteries	Fine fibrosis of liver. Subgranular kidneys. Emphysema. Scars of ulcers in stomach.
53	M	67	Cellulitis of leg	—	Abundant, but not severe	Firm smooth liver. Subgranular kidneys. Emphysema.
54	M	77	Granular kidneys. Arterio-capillary fibrosis	—	Atheroma and calcification of coronaries. Yellow atheroma in aorta	Slightly granular firm liver. Severe osteoarthritis deformans.
55	F	55	Pernicious anæmia	Broad opaque markings in capsule of liver along lymphatics (?)	—	Emphysema. Firm liver, spleen, and kidneys.
56	M	58	Phthisis	—	Slight	Firm liver, spleen, and kidney. Partly descended testis. Bony plates in falx cerebri.
57	F	65	Chronic myocarditis Broncho-pneumonia	Chronic myocarditis	Widespread atheroma in aorta. Atheroma and calcification in coronaries	Subgranular kidneys.
58	M	64	Pleurisy and pericarditis	—	Extensive, but slight	Firm liver, spleen and kidneys.
59	M	34	Disseminated tuberculosis	Small prominent yellow nodules in ascending aorta	—	Aortic ring thickened. Abundant fine grooving of calvarium.
60	F	47	Phthisis	Scars on face, partly radiate. Decided thickening of aortic ring	—	Granular cirrhosed liver. Large red subgranular kidneys.
61	M	51	Aneurism of arch of aorta	—	Slight in descending aorta	Tough liver. Subgranular kidneys. Spondylitis deformans.
62	F	20	Granular kidneys. Pyelo-nephritis	Thickening of aortic ring. Yellow atheroma on mitral valve (?)	—	Left kidney atrophied. Patchy thickening of aortic and mitral valve.
63	F	59	Strangulated hernia	—	Slight, but widespread. Calcified coronaries	Atrophic granular kidneys. Emphysema.

No.	Sex	Age	Cause of Death.	Signs of Syphilis.	Atheroma not Typical.	Fibrosis of Organs, and Notes.
64	M	40	Gouty pericarditis	—	—	Granular kidneys. Firm liver and spleen.
65	M	39	Appendicitis	Small prominent yellow dots and lines of atheroma	—	Smooth firm pliant liver. Intense emphysemas.
66	M	46	Progressive anæmia	Arborescent white lines in epicardium (?)	—	Emphysema. Smooth firm liver, spleen and kidneys. Malformed foot.
67	F	45	Phthisis	—	—	Atrophic granular kidneys. Fibrofatty liver.
68	M	27	Chronic ulcerative endocarditis	—	Slight	Firm nutmeg liver.
69	M	19	Aortic regurgitation from rheumatic endocarditis	Congenital syphilis. Yellow lines of atheroma in descending aorta. Fibroid patches in anterior mediastinum. Slight pulmonary atheroma. Tiny gummata under capsule of liver	—	Large subgranular kidneys.
70	M	52	Dysentery. Abscess of liver	—	—	Subgranular kidneys.
71	M	42	Cirrhosis of liver. Hæmatemesis	Gummata and fibrosis in testes, liver, lung, and near pancreas	—	Firm pliant liver, spleen and kidneys.
72	M	50	Peritonitis, suppurative	Fibroid plates in pleura and peritoneum (?)	—	Sacculated colon. Stricture in appendix. Pliant firm malformed liver. Firm kidneys.
73	F	36	Phthisis	—	—	Atrophic granular kidneys. Fine fibrosis of liver.
74	M	37	Phthisis	—	—	Cavernous angiomata in liver.
75	M	35	Pericarditis	Atheroma with great thickening in descending aorta	—	Granular kidneys. Dropsy.
76	F	45	Softening of brain from multiple arterial thrombosis	Deep vascular grooves in calvarium. Mitral stenosis. Lines and dots of yellow atheroma in descending aorta. Fibrous node in substance of liver. Two calcified nodules on spleen	—	Subgranular kidneys. Smooth pliant liver.
77	F	40	Leukæmia	—	—	Firm pliant liver.
78	M	67	Bronchitis. Bronchiectasis	General atheroma and dilatation of ascending aorta and arch. Atheroma of pulmonary artery	—	Spondylitis deformans. Emphysema. Firm kidneys with cysts. Large prostate.
79	M	71	Cirrhosis of liver. Large granular kidneys	—	Pipestem coronary arteries	Calcification of aortic and mitral rings. Enlarged prostate. Chronic ulcer of stomach.
80	F	55	Pneumonia	Gummata in liver and in pelvis. Linear streaks of atheroma in abdominal aorta	—	Fine fibrosis of liver.
81	F	77	Uræmia	All stages of atheroma (?)	—	Subgranular kidneys.
82	F	42	Pleuropneumonia and pericarditis	—	—	—

No.	Sex	Age	Cause of Death.	Signs of Syphilis.	Atherom not Typical.	Fibrosis of Organs, and Notes.
83	M	26	Phthisis	—	—	—
84	M	59	Fracture of skull. Abscess in cerebellum	—	Extensive atheroma and calcification in abdominal aorta and iliacs	Firm smooth liver. Paraduodenal fossa.
85	M	71	Senile gangrene	All stages of atheroma (?) Patchy thickening of capsule of spleen	—	Tough kidneys. Partly descended testis.
86	F	36	Phthisis. Perforation of ulcer in intestine	—	—	Smooth tough pliant liver. Subgranular kidneys.
87	M	69	Prostatectomy	Most severe atheroma in all stages. Gummatous fibrosis of pleura. Calcified nodule in spleen	Atheroma of coronaries	Smooth firm pliant liver
88	M	67	Phthisis	All stages of severe atheroma (?)	—	Arteriosclerosis. Firm pliant kidneys, with patchy atrophy.
89	M	57	Syphilitic arteriosclerosis	All stages of atheroma with thrombosis. Deep vascular grooves in calvarium. Bony nodes on tibiæ.	—	Subgranular kidneys. Fine fibrosis of liver. Emphysema, osteoarthritis and spondylitis.
90	M	52	Cerebral apoplexy	Thickened contracted aortic valves (?)	Slight	Large tough irregular kidneys. Large hobnailed liver. Chronic and subacute endocarditis. Emphysema.
91	M	51	Uræmia	Calcified plates in old pericardial adhesions. Great thickening & stenosis of mitral valve (?)	—	Atrophic granular kidneys. Small smooth tough liver.
92	F	38	Puerperal peritonitis	—	Slight	Subgranular slightly lumpy kidneys.
93	M	40	Bronchopneumonia, pleurisy, peritonitis	—	—	Chronic and subacute nephritis.
94	M	53	Gouty kidneys	—	—	Firm smooth liver and spleen. Atrophic granular kidneys.
95	M	55	Empyema	Calcification of old pleural adhesions. Fibrosis of lung with bronchiectasis (?)	Slight	Slight fibrosis of liver and kidneys.
96	F	34	Puerperal peritonitis	—	—	—
97	F	59	Pleuropneumonia and pericarditis	—	—	Chronic nephritis.
98	F	59	Myocarditis, &c.	Syphilitic endarteritis, endocarditis and myocarditis. Atheroma in all stages.	—	Subgranular kidneys.
99	M	60	Phthisis	—	Slight	Kidneys subgranular, tending to atrophy.
100	M	79	Arteriosclerosis, dropsy, erysipelas	All stages of atheroma in aorta Atheroma and thrombosis of pulmonary artery. Perihepatitis with false capsule	—	Granular or lumpy kidneys. Multiple ulceration of stomach. Enlarged prostate.

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